The Meixner Test

TO THE EDITOR: I would like to comment on the "Mushroom-Related Call Data Form,"[1(p288)] in the October article, *"Amanita phalloides*-Type Mushroom Poisoning," which gives directions for performing a Meixner test. As one of the authors of the reference cited (number 9),[1(p289)] I strongly suggest that you change the instruction "add two to three drops of concentrated hydrochloric acid" to "add a single drop of concentrated hydrochloric acid" since it is imperative to use the smallest amount of acid possible. Even two drops will cut down on the detection limit of low amounts of amatoxins such as those found in certain *Lepiota* and possibly *Galerina* spp. Personally, I prefer to use a microhematocrit capillary tube which seems to deliver just about the right amount of acid. Also, I always spot a "control" drop of acid adjacent to the mushroom extract being tested. This is important because some papers may produce color reactions for some unknown reason. Minute quantities of acid are also essential when testing remnants of the food, stool or vomitus. The latter two may contain visible amounts of the toxins when tested within approximately 15 hours after ingestion. Stool and vomitus should be diluted with methanol, centrifuged and filtered. The filtrate can be spotted on newsprint. Methanol will help to extract the toxins.

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REFERENCES

1. Olson KR, Pond SM, Seward J, et al: *Amanita-phalloides*-type mushroom poisoning. West J Med 1982 Oct; 137:282-289

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Dr Olson Replies

TO THE EDITOR: We appreciate Mr Vergeer's comments regarding the Meixner test, and regret the error in the amount of hydrochloric acid to be used which appeared on the data form.

We would like to remind readers that even if the *test* is truly negative, it does not necessarily rule out *ingestion* of amatoxins. Mixtures of mushrooms may be consumed and the one that is presented for testing may not be the toxic one.

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Anaphylaxis Following Zomepirac Ingestion

TO THE EDITOR: This is a report concerning anaphylaxis following the ingestion of zomepirac sodium (Zomax).

In a middle-aged man who was taking antihypertension medications, low back pain developed while he was exercising. Ingestion of a 100 mg tablet of zomepirac was followed in ten minutes by substernal oppression, diaphoresis, fecal and urinary incontinence, collapse, wheezing, cyanosis, pruritus and urticarial rash. The patient said that he had had an uneventful two-week course of zomepirac therapy ten months before. Tests were negative for asthma or aspirin allergy. There was prompt response to administration of saline and steroids, with an uneventful recovery.

There are two cases reported in the literature.[1,2] There have been no reported cases to the Arizona Poison Control Center (personal communication, Dr T. G. Tong, January 4, 1983). A medical director from the manufacturer said he was aware of only a few cases of anaphylaxis (personal communication, Dr J. D. Siegfried, January 5, 1983). A staff person from the Food and Drug Administration stated that there was quite a large number of similar cases but he could not cite a figure (personal communication, Mr R. A. Eaton, Division of Drug Experience, January 5, 1983).

The recent announcement by the manufacturer that there have been more than 1,000 cases of anaphylaxis and five deaths seems to belie the paucity of cases reported in the literature. Perhaps physicians should be more conscientious in reporting such observations more promptly.

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1. Samuel SA: Apparent anaphylactic reaction to zomepirac (Zomax) [letter]. N Engl J Med 1981 Apr 16; 304(16):978
2. Corre KA, Rothstein RJ: Anaphylactic reaction to zomepirac. Ann Allergy 1982 May; 48:299-301

Lasègue, Not Laséque

TO THE EDITOR: Lasègue, not Laséque.

In the Epitome section of the November 1982 issue, the famous French physician's name was misspelled in two ways, namely with an accent *aigu* instead of an accent *grave* and with a "q" instead of a "g."[1]

Should one trust the contents of a bottle if the label is wrong?

ERNST W. BAUR, MD
Tacoma, Washington

REFERENCE

1. Purcell GA: Spinal stenosis, *In* Important advances in clinical medicine—Orthopedics (Epitomes of Progress). West J Med 1982 Nov; 137:412

The Use of Acronyms

TO THE EDITOR: Like Dr Alfred Robinson,[1] I, too, am irritated by the use of acronyms. I have made my complaints to the various publications I receive indicating the confusion it causes and the increased difficulty in trying to digest the articles I read that are "salted" with these acronyms. Please tell me why they are used.

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REFERENCE

1. Robinson AG: Acronyms in medical papers (Correspondence). West J Med 1982 Sep; 137:251

EDITOR'S NOTE: We entirely agree with complaints against the use of acronyms in medical journals. In copyediting, *WJM* staff eliminate most of the acronyms originally appearing in accepted manuscripts. How-

ever, occasionally a term being replaced is so long or is used so frequently throughout a paper that the decision is made to let the acronym stand and spell it out in an "Abbreviations Used in Text" box at the beginning of the article. Nonetheless, we appreciate it when readers call attention to the overuse of acronyms and let us know that we are slipping into "indigestibility." —MSMW

Steroid Therapy and the Risk of Gastrointestinal Injury

TO THE EDITOR: Pezner and Lipsett[1] suggest that while corticosteroids are highly effective in patients with metastatic disease to the brain, the use of dexamethasone in dosages of 12 mg or more per day increases the risk of peptic ulcer disease (PUD). Major flaws in this study's method make it unreasonable and potentially dangerous to accept this conclusion.

In this series, PUD developed in five patients who received "high dose" steroids; 84 patients also received similar high doses but PUD did not develop. Seventeen patients did not receive at least 12 mg per day, and in none of these patients did PUD develop. These 17 patients make up the control group (unidentified by the authors), on the basis of whose comparison with the other 89 (treatment group) the authors base their conclusions.

It is in general difficult to prove cause-and-effect relationships in retrospective studies, particularly when groups being compared are not shown to be similar in baseline characteristics. If we are to believe that the use of a certain dosage of steroids is the independent variable associated with the development of PUD in these patients, we must first be assured that there are no other independent variables, such as differences in age, type and degree of underlying disease, other modes of treatment and the like. Not only is none of this information clearly available about the two groups in this series, but there is at least the suggestion that patients who received the higher doses had more severe illness than those who did not. We are not told anything about the use of other medications or the presence of other significant diseases in either of the groups in general, but we are told that four of the five patients in whom PUD did develop had seven other plausible causes for this complication, not including their underlying central nervous system disease. Finally, while the authors claim that the so-called relationship between steroid use and PUD was dependent upon the dose of dexamethasone used, "tapering of dexamethasone dosage had been started in two patients before the peptic ulcer disease developed . . . ." (We are not even told whether their total dosages were below 12 mg per day at the time of onset of their symptoms.)

Of even greater concern is the misuse, or rather nonuse, of statistical analysis in this paper. The authors state at the end of their Methods section that statistical significance was tested by the x^2 method, but in fact they do not at any point in the paper make any statistical comparisons. In fact the difference between the treatment and control groups with regard to development of PUD is not statistically significant. Five of 89 is easily seen to represent just *under* 1 in every 17 patients, so the absence of any PUD in the control group of 17 is intuitively well within the realm of chance statistical variation (even if both groups were in fact matched with regard to all variables except steroid use, and if treatment entailed no increased risk of PUD). Not surprisingly, x^2 testing shows the difference between the groups to be far from significant, with a P value of close to 0.5.

There may be some point in reporting a retrospective review of complications seen in a group of patients with brain metastatic disease, most of whom received at least 12 mg per day of dexamethasone therapy; it is irresponsible, on the other hand, to state conclusions that are not only impossible to evaluate because of the incompleteness of the information presented, but which even in the best possible case are not supported by the limited data presented. It is furthermore dangerous to do so when misinterpretation of such data, as in the authors' discussion, might lead some readers to withhold an extremely valuable medication.

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REFERENCE

1. Pezner RD, Lipsett JA: Peptic ulcer disease and other complications in patients receiving dexamethasone palliation for brain metastasis. West J Med 1982 Nov; 137:375-378

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TO THE EDITOR: The fine article by Pezner and Lipsett, "Peptic Ulcer Disease and Other Complications in Patients Receiving Dexamethasone Palliation for Brain Metastasis," discusses the association between corticosteroid therapy and gastrointestinal injury.[1] They also raise important questions regarding the use of prophylactic antacids in patients receiving high doses of dexamethasone and other steroids. I would like to add some comments to their discussion.

Theoretically, corticosteroids have significant ulcerogenic potential. It is unlikely, however, that dexamethasone alone (at doses higher than 12 mg per day) was responsible for the development of peptic ulcers in the five patients described in the study. Three of the five patients were also using unspecified doses of nonsteroidal anti-inflammatory agents (NSAIA's), two patients had thrombocytopenia and one patient had a history of ethanol abuse. These associated factors undoubtedly increase the risk of peptic ulcer disease and gastrointestinal bleeding developing.

Whereas nonsteroidal anti-inflammatory agents have well-documented potential for causing gastrointestinal injury, controversy concerning the association of corticosteroid treatment and peptic ulcer disease remains